Pathogenesis of the Vertical Root Fracture

6

Richard E. Walton and Eric Rivera

Abstract

When a vertical root fracture reaches the outer surface of the root, it communicates with the periodontal ligament, and an inflammatory process begins in this area. On communication with the oral cavity through the gingival sulcus, foreign material and bacteria obtain access to the fracture area. The inflammatory process increases with a slow separation of the fractured parts of the root and a breakdown in the periodontal ligament and the alveolar bone. Consequently, granulomatous tissue is formed, and bone subsequently resorbs with typical features such that most are clinically manifested. This chapter will describe the histopathological features of the hard and soft tissues associated with vertical root fractured teeth, including the various tissues and elements involved.

Introduction

A vertical root fracture (VRF) is not an uncommon complication in root canal–treated teeth [1, 2]. This results in major damage to the periodontium. There is substantial clinical evidence that this vertically aligned fracture also generates primarily a vertical destructive lesion of the supporting structures [3, 4]. This damage includes both the soft tissues and the adjacent alveolar bone [5]. Destruction may occur slowly but is often rapid and profound. The clinical signs, symptoms, and findings are such that a periodontal disease-type lesion is often a first impression

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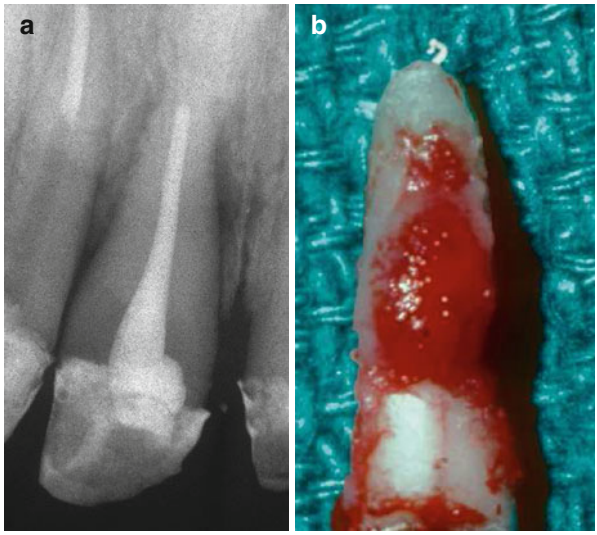

Fig. 6.1 A deep probing defect on the buccal aspect of the root in an endodontically treated maxillary central incisor. The radiograph (**a**) shows lateral radiolucencies in the mesial and distal aspect of the root. The extracted tooth (**b**) shows the inflammatory tissue attached to the root

[6]. When a VRF is diagnosed clinically, the clinical evidence is that the fractured root cannot predictably be repaired and salvaged [7, 8].

Observations following flap reflection and/or tooth or root removal as the result of VRF show an inflammatory lesion adherent to the root surface directly overlying the fracture (Figs. 6.1 and 6.2).

Why is the destruction so profound? Currently, there is one published study [9] that examined fractured roots and adherent tissues histologically. Specimens were studied to ascertain the pattern of the fractures and to clarify the nature and the location of irritants that were associated with the fracture. In addition, the inflammatory lesions were examined as to the nature and pattern of inflammation. These findings from this study [9] are the primary basis of information for this chapter. In this study [9], roots with clinically identified fractures were obtained following tooth extraction or during exploratory surgery (Figs. 6.3 and 6.4).

More about the surgery flap procedure as a clinical adjunct to help diagnose VRF is described in Chapter 4.

The specimens were fixed in formalin, decalcified, embedded in paraffin, and cross sectioned. Histological sections were stained with H&E to identify general characteristics; alternate sections were stained for bacteria. Regions studied with the light microscope were from the cervical, middle, and apical thirds.

The histology showed patterns of the fractures in the root. Also demonstrated was that the canal and fracture spaces contained combinations of irritants that were etiologies for the inflammatory lesions that overlaid the root surface.

The characteristics of the fractures were important and followed a general pattern but with variations. These types are demonstrated in Chap. 2 on categorization.

All were in a buccolingual plane. Most extended to both surfaces (complete fractures), but some were to one surface only (incomplete fractures) (Figs. 6.5a–c and 6.6a, b).

All the fractures communicated with a canal or canals. Most fractures were likely "old" because they contained an ingrowth of vital tissue. Another indicator that the

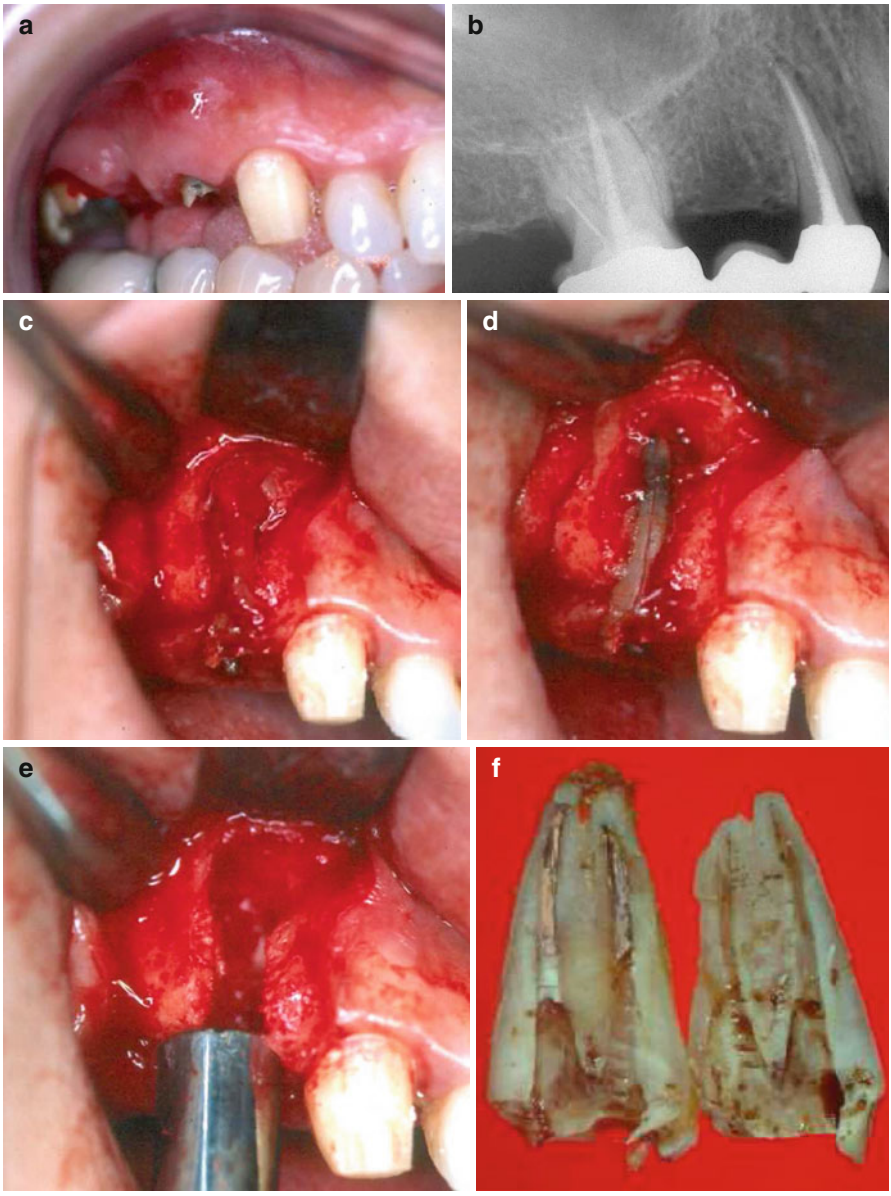

Fig. 6.2 (**a**–**f**) Patient presented to the dental office with a complaint of a "loose bridge" and "suppuration from the gingivae." The maxillary first premolar was used as an abutment together with the maxillary canine (**a**). The probing defect was not contributory. The periapical radiograph (**b**) revealed widening of the PDL on the mesial aspect of root. Since the diagnosis of VRF was not conclusive, it was decided to perform surgical flap procedure for diagnosis and treatment. When the flap was performed, a large bony dehiscence was seen (**c**) filled with granulation tissue. After removal of the inflammatory tissue, a VRF was seen from the coronal part to the apical (**d**). The dehiscence of the buccal bone which was facing the fracture can be seen very clearly (**e**). The fracture was a typical buccolingual fracture, and the root was extracted in two parts (**f**) (Courtesy Prof. A. Tamse)

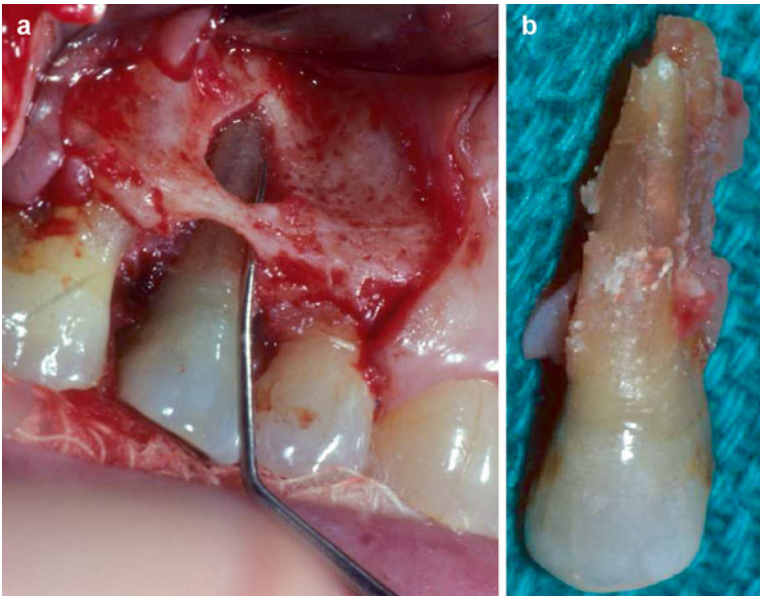

Fig. 6.3 (**a, b**) A large fenestration can be seen upon flap procedure performed on a maxillary lateral incisor (**a**). The inflammatory tissue can be seen attached to the fractured root (**b**)

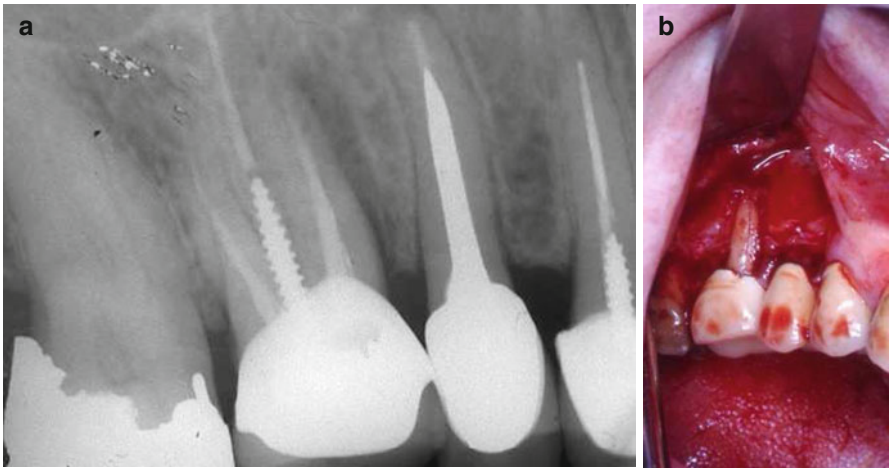

Fig. 6.4 (**a**, **b**) Patient's chief complaint in this case was "I have an abscess that comes and goes for nearly a year." The tooth was endodontically treated 4 years earlier and a crown placed. Upon examination, a 10 mm probing defect was measured in the mesiobuccal aspect. The radiograph (**a**) shows a previously treated maxillary first molar and a large lateral radiolucency along the mesiobuccal root. Since there was no sinus tract and VRF diagnosis was inconclusive, a surgical flap procedure was performed (**b**). A complete bony dehiscence can be seen which was the result of a long-standing inflammation in the area facing the fracture (Courtesy Prof. A. Tamse)

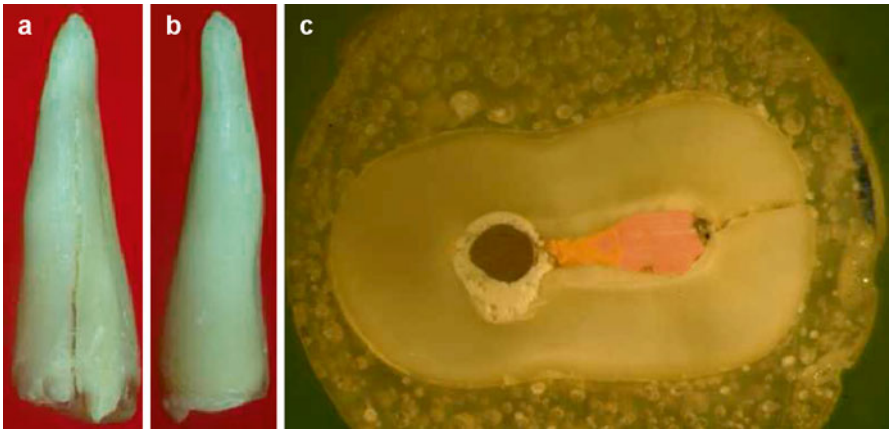

Fig. 6.5 An incomplete VRF in an extracted maxillary premolar due to a VRF. The fracture can be seen in the buccal aspect of the root (**a**) but not in the palatal one (**b**). Cross section of the root (**c**) demonstrates the incomplete fracture from the root canal to the external buccal surface (Courtesy Prof. A. Tamse)

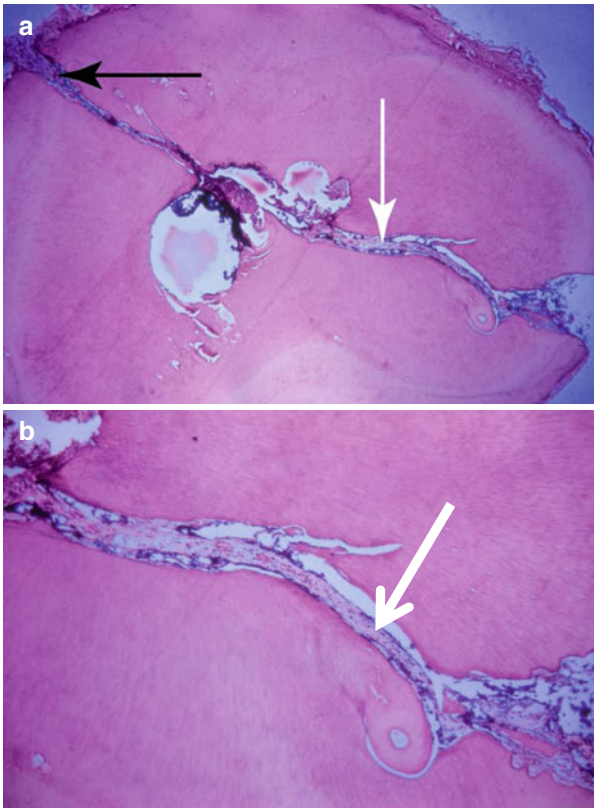

Fig. 6.6 (**a**, **b**) A histological section of a vertically fractured single-rooted maxillary premolar showing the complete buccal to palatal fracture. Areas of resorptions and appositions of bone can be seen along the fracture border with vital tissue penetrating between the fragments. See the *white* and *black arrows* (**a**, **b**), These are an indication that the fracture had occurred in the past

Fig. 6.7 VRF in mesial root of a mandibular molar. Although a very wide separation of the segments can be seen, it is due to an artifact. A complete buccolingual fracture is evident. Colonies of eosinophilic bacteria (red stained) are visible on the fracture surface (*box insert*). Sealer and gutta-percha are black because they block transmitted light and can be seen throughout the canal (Brown and Brenn. Mag ×60)

fracture had occurred in the past was resorptions and appositions of cementum-like tissue on the walls of the fracture (Fig. 6.6). The contents of the fractures were generally associated with potential and actual irritants. Bacteria were always present (Fig. 6.7).

These bacteria were often in a biofilm form or within tubules. Necrotic tissue was evident, and foreign debris such as food remnants were an occasional finding. Sealer and/or gutta-percha were also often observed. The root canal contents were similar to the fractures. Bacteria were always present, often as a biofilm and within tubules. Many areas of the canal contained necrotic tissue and amorphous debris, sealer, and gutta-percha. Inflammation was always present on the root surface and overlying the fracture. The characteristics of the inflammation were similar to the periapical granuloma, that is, predominantly chronic inflammation. The lesions were bacteria free.

The interpretation of the histological findings is that the VRF is a dynamic entity with a unique microenvironment of tissue destruction. The fracture itself resembles a *long* apical foramen that communicates with a canal space that contains numerous potential and identifiable significant irritants. These irritants percolate through the fracture to the surface. There, these irritants contact connective supporting tissues and induce inflammation similar (or identical) to what occurs at the apex. The irritants are nonspecific and/or antigenic, thereby resulting in an immune response [10]. The outcome is both direct and indirect tissue damage and destruction of periodontium (both soft and hard tissues) in the region of the fracture.

The inflammation, as stated above, resembles the periapical granulomatous response. It is established [10] that the primary source of irritant that induces this response is necrotic tissue that contains bacteria. So it is not surprising that periapically and laterally, their histological appearance is similar. The lateral root surface lesion includes a predominance of chronic inflammatory cells (Fig. 6.8) and an absence of bacteria.

However, the bacterial colonization and biofilm formation within the canal is important in a pathogenesis of tissue destruction following VRF. Although specific bacterial species have not been conclusively identified in the fractured root, they are

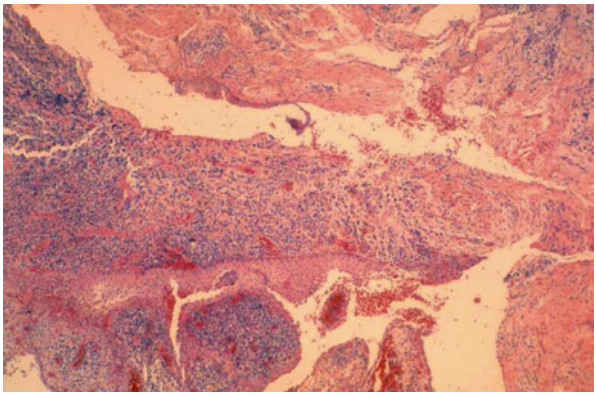

Fig. 6.8 Inflammatory tissue attached to the lateral surface of a vertically fractured root. There is a predominance of chronic inflammation and an absence of bacteria (Courtesy Prof. A. Tamse)

known to be present and important in both initial and in failed root canal treatments [11, 12]. The frequent appearance of a biofilm of bacteria (Fig. 6.7 box insert) is important. Biofilms are a particularly potent irritant [13]. Biofilms tend to persist and are composed of mixed flora that includes pathogenic bacteria [14]. Gram stain showed the presence of gram-positive microorganisms; these are a pathogen associated strongly with periapical pathosis.

Although the sources of these bacteria within the fracture have not been identified, they could arrive by different avenues [15]. These avenues would include from the oral cavity directly into the fracture [16] and via the periodontium or from the remnants of bacteria not removed during root canal treatment [17, 18].

In addition to bacteria, other potential and actual irritants likely are significant contributors. These include food debris, sealers, necrotic tissue, and other possible contaminants such as saliva or other chemicals present in the oral cavity. All these would have direct access to the periodontal tissues via the fracture. Similar to the necrotic pulp space, the defense mechanisms have no or limited access to the fracture space. The finding that the fractures demonstrated a variety of patterns is interesting as well as clinically significant. Different patterns were noted on the extracted teeth as well as histologically. These variations have been reported in other studies [19, 20]. Although not determined, those incomplete fractures likely demonstrate inflammatory lesions that reflect the fracture. Therefore, a probing defect may not be present when the fracture and associated inflammation is limited. If the fracture is only on the lingual, it would not be visible with flap reflection on the facial. If the fracture does not extend to the cervical margin, this may explain why many VRFs do not have associated probing defects (See additional information in Chap. 4 on diagnosis of VRF) (Fig. 6.9).

Importantly, the pathosis associated with the VRF is neither true periodontal disease nor is it a true "combined endo-perio" lesion. There was no histological evidence of a loss of attachment, which, in addition to bone resorption, is a feature of periodontal disease [21]. The inflammatory lesions were attached and adherent at all levels.They represent endodontic pathosis; a probe would pass easily into the inflammation.

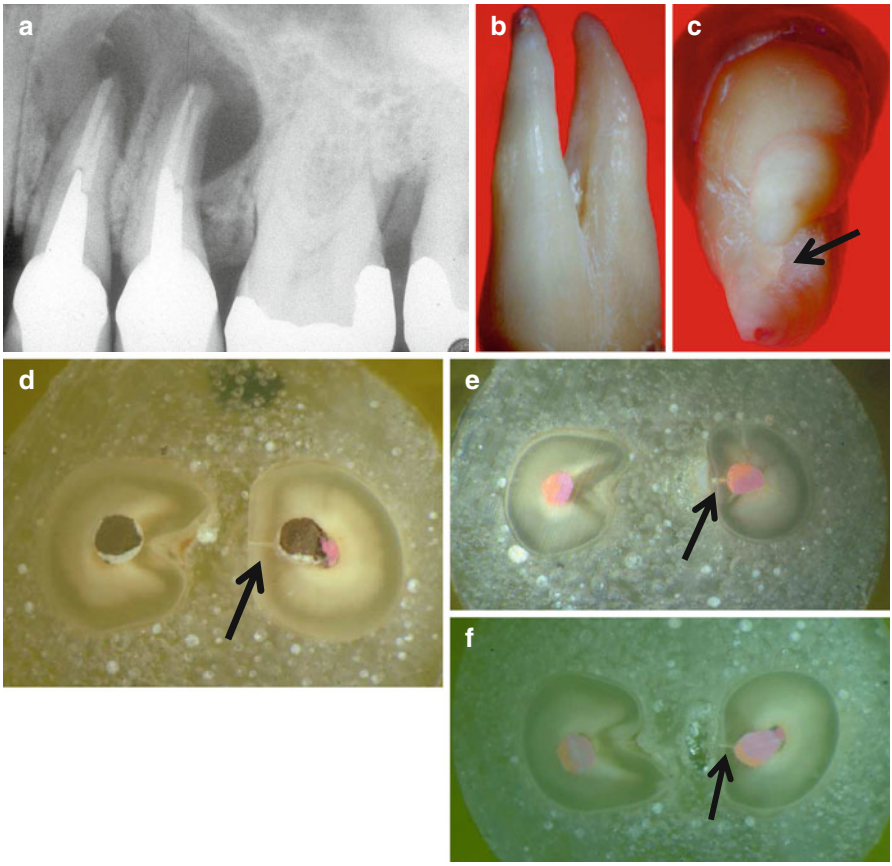

Fig. 6.9 (**a**–**f**) A patient presented to the dental office with a chief complaint of "loose teeth that were treated many years before and a strange discomfort upon touching the gum on the palate." The teeth were endodontically treated and restored with two cast dowels and PFM crowns 11 years previously. Dental examination revealed slight mobility of the two maxillary premolars (**a**). A 7 mm probing defect was recorded in the first premolar, but the probing was normal in all aspects of the second premolar. The attached gingiva in the palatal aspect of the second premolar was sensitive to palpation. The periapical radiograph shows a large radiolucent area in the bone surrounding the two roots and extending mesially to the lateral aspect of the canine and distally to the mesiobuccal root of the first molar. (**a**) The two teeth were suspected of having fractured roots. However, since retreatment prognosis in these teeth was poor, they were extracted. The extracted second premolar is shown in (**b**) In the mesial view of the extracted bifurcated premolar (**c**), a VRF can be seen in the bifurcation aspect of the palatal root (*Black arrow*). Three cross section slices of this root (**d**–**f**) are showing the incomplete VRF in the palatal root (*Black arrows*). No fracture is seen in the buccal root. Note the very minimal remaining dental thickness between the gutta-percha-filled palatal canal and the external surface of the root facing the bifurcation area (Courtesy Prof. A. Tamse)

Conclusions

The pathogenesis of the VRF has been demonstrated in the histological examination of cross sections of extracted roots. Both the fractures and the canals with which they communicated contained irritants capable of causing or contributing to the inflammatory lesion on the root surface. Fractures are not always complete buccal to lingual or coronal to apical but contained tissue, bacteria and root filling materials, necrotic debris, and other nonspecific irritants. Canals are similar in that the same irritants can be demonstrated. The interpretation is that the fracture is a long apical foramen communicating with spaces that contain profound irritants that generate an immune/inflammatory response that significantly damages the supporting periodontium.

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